leukocytosis is out of the question, and the condition in our opinion can only be classed among the leukemias, and in view of the presence of the young elements mentioned, the name promyelocytic leukemia is in order.

The significance of the eosinophilia in the case of mediastinal and pituitary tumor is of statistical value only. The origin of the eosinophiles, or rather the stimuli that are responsible for their formation, is still a question to be answered by the experimental hematologists. The interesting work of Opie on this subject may throw some light upon their nativity as well as upon the substance from a chemical point of view, necessary for their increase in the peripheral blood.

A CASE OF BANTI'S DISEASE: SPLENECTOMY FOLLOWED BY TYPHOID FEVER AND APPENDICITIS.

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A considerable number of cases of Banti's disease have already been published and numerous reports of splenectomy performed for the relief of the symptoms of this disease are on record and favorable results have been ascribed to this operation. Although patients who have suffered from this disease and have been apparently cured by splenectomy remain in good health for years, there is hardly anything known of the behavior of these splenectomized individuals during attacks of infectious diseases.

The case of Banti's disease which I wish to present was successfully treated by splenectomy and passed through an attack of typhoid fever one year and a half after the operation. During the course of the typhoid it was necessary to remove the appendix owing to the occurrence of a suppurative appendicitis. It is chiefly on account of the reaction of the splenectomized patient to these infectious diseases that sufficient interest is attached to this case to justify recording it. The existence of syphilis in this case, as shown by the positive Wassermann reaction and by other evidences pointing to hereditary lues, is also of importance.

The following is a brief history of the case, which was referred to me through the courtesy of Dr. William E. Beardsley.

L. G. H., aged seventeen years, female; native of the United States, of German parentage; single, no occupation; admitted to the Jewish Hospital of Brooklyn on January 25, 1909; discharged February 7, 1909. Family history does not offer any significant facts. She had measles, mumps, and whooping cough in childhood, and some eye trouble three years prior to admission to the hospital; no alcoholic history; only menstruated once eight months before admission and never since. Her present illness dates back five years. She noticed a mass in the upper portion of the abdomen, which was painful at times, especially on pressure, which continued to increase in size. At the same time, she noticed a progressive weakness and loss of energy. She suffered from dragging sensations in the abdomen, slight jaundice and itching, and occasional attacks of epistaxis.

The physical examination showed an undersized, imperfectly developed girl; sclera of slight yellowish tinge; conjunctiva pale; pupils equal and reacting normally to light and accommodation. An examination of the throat did not show anything abnormal; the tongue was moist and clean. The heart was normal to percussion; apex beat was not visible nor palpable; there was accentuation of the second pulmonary sound and a clicking second sound at the apex. The pulse was rapid, soft, and easily compressible. There was dulness from the level of the tenth dorsal vertebra on both sides posteriorly and diminished tactile and vocal fremitus and diminished respiratory sounds. The liver dulness reached from the upper border of the fourth rib to 7½ cm. below the costal margin at the mammary line. The lower border of the liver was easily palpable at that location. The lower border of the spleen was palpable about 9 cm. below the free costal margin. Its notch could be felt two fingers' breadth from the median line, about 6 cm. below the costal arch. The whole abdomen was distended and a diastasis of the recti muscles allowed a mass the size of an orange to protrude just below the ensiform cartilage. The submaxillary glands and the inguinal glands were very slightly enlarged. There was a slight icterus. The hair in the axillæ and around the pubes was scant. The reflexes were normal. There was some ascites, but no edema of the lower extremities. The urine was free from albumin, sugar, and casts; acid; specific gravity 1010. The feces showed nothing abnormal. The Wassermann reaction was strongly positive. The test was made by Drs. Noguchi and Lintz.

The blood examination showed: Red blood cells, 3,200,000; hemoglobin, 48 per cent; leukocytes, 5500; polynuclears, 61 per cent.; lymphocytes, 39 per cent.; eosinophiles, less than 1 per cent.

The specific treatment, which was thorough, and included intramuscular mercurial injections, was not followed by any favorable results, nor was the application of the *x*-rays and the administration of the arsenical preparations of any benefit to this patient.

The temperature curve during her two weeks' stay in the hospital showed only slight rises, reaching at times to a little over 101°. Her pulse varied from 76 to 110, her respirations from 20 to 35 in an irregular way.

The condition of the patient becoming gradually worse, splenectomy was decided upon and was performed by Dr. Walter C. Wood, at the Brooklyn Hospital, on April 26, 1909. The spleen weighed 1 pound 11 ounces, and measured 8 by 4 by 3 inches. It was of moderately soft consistency; there was an area 1½ inches in width at its upper portion, which was soft and semifluctuating. There were numerous whitish areas, which on section showed a cheesy appearance. A microscopic examination showed caseation and giant cells.

From the time of the operation improvement was rapid and continuous, and when she came under my observation again, on September 26, 1909, she appeared to be in a decidedly improved condition. The blood examination the following day showed: Red corpuscles, 5,300,000; hemoglobin, 68 per cent.; leukocytes, 12,800; polynuclears, 58.3 per cent.; lymphocytes, small, 35.5 per cent.; large, 0.04 per cent.

On December 12, 1909, the blood count was: Red corpuscles, 6,160,000; hemoglobin, 65 per cent.; leukocytes, 11,400; polynuclears, 61.5 per cent.; lymphocytes, small, 35 per cent.; large, 2.1 per cent. The eosinophiles were always less than 1 per cent.

There was, therefore, a very remarkable improvement in the condition of the blood, only the percentage of hemoglobin remained comparatively low, while the number of red corpuscles came up to and slightly above the normal limit. The leukocytes increased decidedly in number and came rapidly up to the normal percentage. The relative proportion of the polynuclears to the lymphocytes did not show any marked changes before and after the splenectomy; the eosinophiles were not increased in number.

The patient remained in apparently good health until October 1, 1910, when she was taken with typhoid fever and was placed under my care at St. Catherine's Hospital on the eleventh of that month. The attack was of medium severity; the Widal reaction was strongly positive. The temperature curve differed in no way from that of a typical typhoid up to the eighteenth day of her stay at the hospital, October 28, when a gradual rise of temperature occurred, also a rise in the pulse and respiration, with the setting in of an appendicitis.

October 13, 1910, two weeks after the onset of the typhoid fever, the blood examination was as follows: Red corpuscles, 4,100,000; hemoglobin, 75 per cent.; leukocytes, 4500; lymphocytes, 21 per cent.; polynuclears, 70 per cent.; mononuclears, 7 per cent.; eosinophiles, 2 per cent.; color index, 0.8 per cent.

On October 28, 1910, the day of the onset of appendicitis, the leukocytes were 9800, of which 68 per cent. were polynuclears. The temperature rose during the next two days from 101.8° to 105.4°, with a corresponding rise of the pulse to 135. Chills, severe pain in the right iliac fossa, with exquisite tenderness, resistance at that location, the anxious expression on the face of the patient, and a rise of the leukocyte count to 15,000 on October 30, pointed to a serious involvement of the intestines at the iliocecal region. The operation, performed by Dr. Frank D. Jennings, revealed a suppurating appendicitis and also a large cystic kidney, which was displaced downward and fixed in its abnormal position. The liver was found smaller than normal and the gall-bladder decidedly paler than usual. The patient made a good recovery, her temperature being of the type of a continua, 103° in the evening to about 102° in the morning, until November 7, when it gradually fell, reaching normal on November 12, the thirty-third day of her stay at the hospital. From then on it showed only slightly rises to about 100, and finally remained normal until her discharge from the hospital on December 11, 1910.

On November 1, two days after the operation, the leukocytes fell to 10,500, the polynuclears being 69 per cent.

By November 16, 1910, the blood count showed: Red blood cells, 4,400,000; hemoglobin, 75 per cent.; leukocytes, 9800; lymphocytes, 25 per cent.; polynuclears, 70 per cent.; mononuclears, 3 per cent.; eosinophiles, 2 per cent.

The patient continued to enjoy good health up to the present time and was examined by me on April 10, 1911. Her weight was 99¼ pounds. There was a slight enlargement of the posterior cervical glands on the right side, but no other glandular enlargement. There were no abnormalities about the chest. The liver dulness began at the sixth intercostal space and reaches down about three fingers' width below the free border of the ribs. The liver felt a little firmer than normal and was smooth. At its extreme right a firm irregular mass could be felt, which was most likely the right cystic kidney as found at the time of the operation. The liver dulness at the mamillary line occupied only two intercostal spaces. The upper and lower middle incisors were slightly notched. Temperature, pulse, and respirations normal. No other abnormalities were found.

Blood examination made on April 7, 1911, showed: Red blood cells, 5,860,000; hemoglobin, 104 per cent.; leukocytes, 11,600; polynuclears, 78 per cent.; mononuclears and transitionals, 2 per cent.; lymphocytes, small, 17.5 per cent.; mast cells, 0.5 per cent. Blood coagulated almost immediately on lobe of ear.

The points which are most evident in this case are: (1) A typical clinical picture of the disease described by Banti, Senator, and others, namely, splenomegaly; the decidedly enlarged liver; the typical blood findings; the slowly progressive exhaustion were well pronounced in this case. (2) The lack of development of the patient, her appearance being that of a child aged twelve years. (3) The positive Wassermann reaction. (4) The rapid improvement in the condition of the blood, with a decided improvement in the hemoglobin following the operation of splenectomy, but not reaching the normal, and remaining practically stationary for at least nine months, varying from 63 to 78, while in the recent examinations it reached even above normal, 104 per cent. (5) The typical characteristic typhoid, differing in no way from that of patients not operated on, with the exception of the blood findings, which showed a rather high leukocyte count for a case of typhoid and a relatively high polymorphonuclear leukocytosis. (6) The reaction to the surgical interference during the course of the typhoid did not present anything characteristic, and her recovery was as rapid as one could expect in a patient not splenectomized.

The internal secretion of the spleen, which according to modern physiologists[1] activates trypsinogen into trypsin, is either supplied in this case by a supernumerary spleen or spleens, or else is effectively substituted, perhaps, by the succus entericus. At any rate, the proteolytic digestion and metabolism is not deranged.

This case further demonstrates that the enlargement of the spleen in typhoid is not essential to the elaboration of immune substances, again assuming that there are no supernumerary spleens.

There is a possibility that hereditary syphilis and tuberculosis of the spleen form etiological factors in this case, as shown by facts previously stated. Evidences of gastro-intestinal diseases could not be obtained. Willi Schmidt's[2] case, recently published, is that of a boy, aged fourteen years, with retarded development, presenting Banti's disease symptoms and having a positive Wassermann reaction, which was successfully treated by salvarsan injections. In 2 out of 3 cases of Banti's disease recently treated at the Jewish Hospital, of Brooklyn, there was found a positive Wassermann reaction.

Marchand[3] and Chiari[4] reported the presence of hereditary lues in some cases of Banti's diseases. Other cases fail to present any evidence of lues. In the carefully observed case of Emmanual Policek[5] there was a negative Wassermann reaction. The pathological condition was designated as chronic inflammation with connective tissue proliferation and the formation of granulomata, closely resembling tubercles. There were, however, no tubercle bacilli found after splenectomy.

The etiology is still obscure. The success of splenectomy places the chief seat of the disease in the spleen. So much can be conceded at present from the considerable number operated upon, to which the one under consideration may be added.

It is also clear that a toxemia emanating from the spleen is responsible for most of the symptoms observed in these patients. In the cases of this disease that came under my observation, the hemorrhages were most readily accounted for by a toxemia. Neither engorgement of the portal system nor direct pressure on vessels through enlarged intra-abdominal glands seemed to offer a satisfactory explanation for the occurrence of these often severe hemorrhages, while the resemblance to those of undoubted toxic origin was apparent. In one of my cases of this disease hemorrhages occurred early, when there was only very moderate splenic enlargement and no evidence of any involvement of the liver. Is it possible that an excess of internal secretion of the spleen is responsible for the occurrence of the symptoms, such as one observes in that of the thyroid gland? Or is this toxic substance formed through the agency of infection? Sippy[6] found that filtered splenic juice obtained from patients suffering from Banti's disease injected into rabbits caused death in a few hours.

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[1] Schiff, Herzen, Gachet, and Pachon.

[2] Münch. med. Woch., March 21, 1911, p. 625.

[3] Ibid., 1903, p. 11.

[4] Folia Hematologica, 1910, ix, 508.

[5] Prager med. Woch., 1902, No. 21.

Sciolla, on the other hand, in a similar experiment with splenic pulp failed to observe any apparent effect. Umber found an increased destruction of the albuminoids in this disease, while Arthur Müller[7] found normal metabolism in his case.

In conclusion, I wish to emphasize the remarkable results following splenectomy in my case; the lasting improvement and the ability to combat severe infections. Also the restoration of the blood to the normal condition as to the formed elements and to a marked increase in its coagulability.

REMARKS UPON SOME PHASES OF THE SPASTIC-PARETIC SYNDROME OF CEREBRAL DIPLEGIA.[1]

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IF we are to make any signal advance in the treatment and prevention of the spastic-paretic syndrome of infantile cerebral diplegia, we must proceed upon the sound anatomical and physiological laws underlying this disease. At the outset it should be emphasized that infantile cerebral diplegia is not a disease entity, but has a clinical basis only.

It has been stated authoratively that there is a description of Little's disease in Audry's treatise on orthopedics, published in 1741, and that Delpech incidentally published a single case in 1728. But the condition must have existed from the remotest antiquity, and there is no doubt that a careful research into the past, both in medical and graphical records, would reveal further evidence of the condition. While this statement might not hold good for Little's disease, *per se*, as an accident of childbirth, we should consider the frequency of spastic states in hysteria of young girls and admit that such cases might easily be confounded with organic disease originating in early childhood. In 1840 Heine, the pioneer worker in poliomyelitis, anticipated Little by giving a good description of the syndrome, which, however, he named "spastic cerebral paraplegia." This discovery in nosography probably passed unnoticed; just as happened when Little, in 1861, published his classic account of the syndrome which now bears his name. Many of the points elaborated by Little had to be rediscovered later; showing that the medical world did not really assimilate Little's work until years after its publication. The reason for this lack is not far to seek. Little's syndrome was looked upon at first as an obstetrical subject; it then became an orthopedic subject; today it is admittedly a neurological subject. It is, however, an excellent illustration of a borderland disease, and these disorders are almost invariably neglected, and viewed from a partisan standpoint. Fortunately several of the most popular German journals consider only these borderland subjects, whereas American medicine has been very slow in adopting this practice, and therefore lacks much in harmonizing and bringing up to date our knowledge of many subjects.

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[6] Amer. Journ. Med. Sci., p. 428.

[7] Münch. med. Woch., 1909, No. 45, p. 2316.

[1] Read before the New York Neurological Society, February 6, 1911.

Some years after Little's publication Erb and Charcot independently described a form of developmental spasticity of spinal origin. The mental state in such cases was, however, normal, hence the cerebrum was supposed not to have been involved. Today we know that Little's syndrome may be due to an intracranial infection, and yet the intellect may be unaffected. On the other hand, the number of cases of spinal spasticity in children recorded in the literature is steadily multiplying. The syndrome follows or accompanies many injuries and diseases of the medulla and its membranes. Naturally, from the beginning much controversy has arisen as to the respective parts played by the brain and spinal cord in these cases. Such a controversy led still to others: What was to be understood by Little's disease? Should it comprise other forms [illegible]ty or spasticity in childhood, including double athetosis and congenital chorea? Should one believe that the brain and spinal cord might be individually injured in labor, with the production of two syndromes?

As intimated above, and as still holds true in our day, some writers could not dispossess themselves of the idea that injury of the brain must be accompanied by idiocy and *vice versa*. In searching the literature of our subject one must bear this obsession constantly in mind. Inasmuch as spasticity was commonly associated with high degrees of mental defect, the account of Little's disease has been much overcharged.

In attempting to explain the spasticity of cerebral diplegia, it is